# Recurrent ventricular tachycardia of non-ischaemic origin

E Ng ChB MRCP D Adlam BCh MRCP
R P Keal MBBS FRCR G A Ng PhD MRCP

*J R Soc Med* 2004;**97**:23–25

Ventricular tachycardia (VT) in the setting of previous myocardial infarction is usually related to scarring or ischaemia. When ischaemic heart disease is not the cause, the management and long-term prognosis are different.

## CASE HISTORY

A man of 64 was transferred to a tertiary interventional centre for coronary assessment. In the previous six weeks he had been admitted twice to his local hospital with VT. On each occasion he gave a history of sweating, nausea and palpitations; there was no associated chest pain, breathlessness or syncope. 11 years previously he had had a myocardial infarction, but the only risk factor for ischaemic heart disease was smoking in the past. On examination, the heart rate was 250/min, without haemodynamic compromise or any other abnormal findings. The electrocardiogram (Figure 1*a*) showed a broad complex tachycardia. Peak creatine kinase was 248 iu/L and troponin I was raised at 2.95 $\mu$g/L. Chest X-ray showed borderline cardiomegaly. On each occasion the patient was successfully cardioverted with an intravenous bolus of 100 mg lidocaine. After the second episode, oral amiodarone was prescribed.

On transfer the patient was in sinus rhythm (Figure 1*b*). Coronary angiography and ventriculography showed no abnormality. On echocardiography, left ventricular size and function were normal but the right atrium and right ventricle were dilated with impaired function. On MRI (Figure 2) the right ventricular free wall appeared thin, with the suggestion of small aneurysmal pockets and fatty infiltration. These findings were consistent with a diagnosis of arrhythmogenic right ventricular dysplasia (ARVD). The patient was treated with a beta-blocker and an angiotensin converting enzyme inhibitor and fitted with an implantable cardiac defibrillator. The amiodarone was discontinued because of previous thyroid dysfunction when taking this drug.

## COMMENT

ARVD is a cardiomyopathy characterized by localized or widespread fibro-fatty infiltration of the right ventricular myocardium. The consequences include right heart failure

**(*a*)**

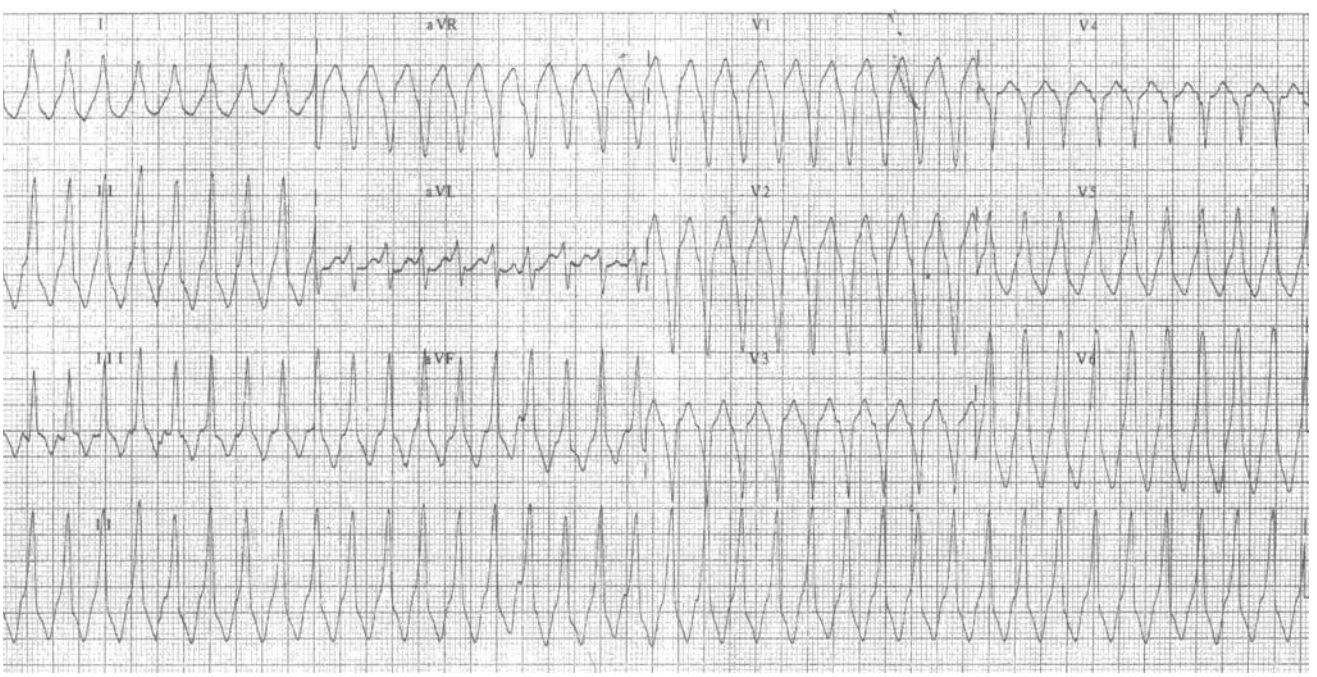
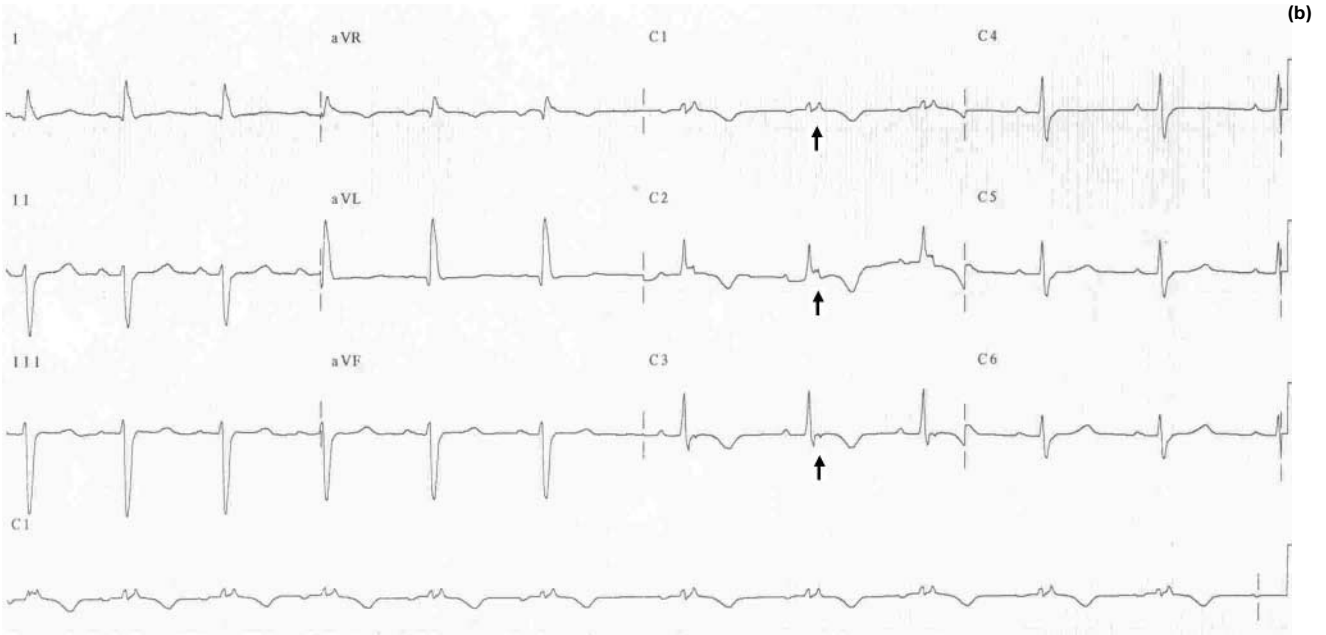



*Figure 1* **Electrocardiograms with and without ventricular tachycardia.** (*a*) Ventricular tachycardia of left bundle branch block morphology and inferior axis. (*b*) Sinus rhythm with partial right bundle branch block, left axis deviation, T wave inversion leads V1–V3 and epsilon waves (arrows)

Department of Cardiology, Glenfield Hospital, Groby Road, Leicester LE3 9QP, UK

Correspondence to: Dr Ernest Ng

E-mail: ernestng321@yahoo.co.uk



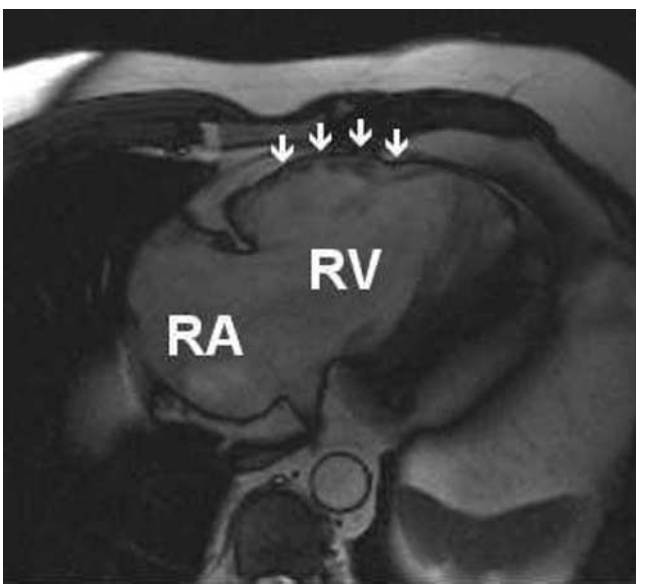




*Figure 2* **Cardiac MRI showing dilated right atrium (RA) and right ventricle (RV) with thinning of the RV free wall.** Aneurysmal pockets (marked) are also seen

and ventricular arrhythmias with left bundle branch block morphology, and risk of sudden death.[1,2] This condition most commonly presents in the second to fourth decades of life and has a male predominance. In some 50% of cases it is hereditary,[3,4] but in the present case there was no family history of heart disease or sudden death.

In this case, the history of myocardial infarction led to an erroneous preliminary diagnosis of VT related to coronary artery disease. The basis of this past diagnosis remains unknown. The patient stated, however, that he had required cardioversion on two occasions during that illness. It was also at this time that amiodarone therapy was initiated—subsequently discontinued because of thyroid dysfunction. It seems possible, then, that this episode was in fact a primary arrhythmic rather than ischaemic event.

The electrocardiographic findings shown in Figure 1*b* are typical, with left axis deviation, partial right bundle branch block, T wave inversion in leads V1 to V3, and post-excitation potentials called epsilon waves.[5] The record made during tachycardia shows a left bundle branch block morphology and inferior QRS axis consistent with an origin near the right ventricular outflow tract. Together with the MRI evidence of right ventricular dysfunction and fatty infiltration of the myocardium[6] these features are diagnostic for ARVD.[4]

The management of ARVD centres on prevention of arrhythmia and sudden death as well as treatment for heart failure. Implantation of a defibrillator allows immediate termination of life-threatening arrhythmia.[7] For refractory heart failure, cardiac transplantation is a final option.

## REFERENCES

1 Marcus F, Fontaine G, Guiraudon G, *et al*. Right ventricular dysplasia: a report of 24 adult cases. *Circulation*. 1982;**65**:384–98

2 McKenna WJ, Thiene G, Nava A, *et al*. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. *Br Heart J* 1994;**71**:215–18

3 Ahmad F, Li D, Karibe A, *et al*. Localization of a gene responsible for arrhythmogenic right ventricular dysplasia to chromosome 3p23. *Circulation* 1998;**98**:2791–5

4 Nava A, Thiene G, Canciani B, *et al*. Familial occurrence of right ventricular dysplasia: a study involving nine families. *J Am Coll Cardiol* 1988;**12**:1222–8

5 Metzger JT, de Chillou C, Cheriex E, Rodriguez LM, Smeets JL, Wellens HJ. Value of the 12-lead electrocardiogram in arrhythmogenic right ventricular dysplasia, and absence of correlation with echocardiographic findings. *Am J Cardiol* 1993;**72**:964–7

6 van der Wall EE, Kayser HW, Bootsma MM, de Roos A, Schalij MJ. Arrhythmogenic right ventricular dysplasia: MRI findings. *Herz* 2000;**4**:356–64

7 Gregoratos G, Abrams J, Epstein AE, *et al*. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation* 2002;**106**:2145–61

# A rapidly increasing pleural effusion

Timothy B L Ho PhD MRCP
Philip Alexander MB BSc Nigel T Cooke MD FRCP[1]

*J R Soc Med* 2004;**97**:25–26

In a patient with pleural effusion, first-line investigations for tuberculosis, if negative, are not conclusive.

## CASE HISTORY

A Rwandan man aged 32 sought advice after two weeks of fever and malaise. He had returned to the UK three months previously from a visit to Uganda during which time he had not taken any malaria prophylaxis. On questioning, he said he had a dull ache in the left upper quadrant of his abdomen and lower back pain. His temperature was 38 °C. There were no focal signs in the chest but a chest radiograph revealed a small left pleural effusion. An ultrasound of his abdomen was unremarkable. Haemoglobin was 13.8 g/dL, white cell count $6.7 \times 10^9$/L, erythrocyte sedimentation rate 63 mm/h. Two blood films for malaria parasites were reported negative. Broad spectrum antibiotics were started. The left pleural effusion was aspirated under ultrasound guidance but no organisms, including acid-fast bacilli, were identified or subsequently cultured. Polymerase chain reaction (PCR) amplification for *Mycobacterium tuberculosis* DNA on the pleural aspirate was also negative. A Heaf test was read after seven days as grade I and a HIV test was negative. Despite ten days of antibiotics the patient continued to have a swinging pyrexia. A chest radiograph revealed that the left pleural effusion now occupied the entire hemithorax. A pleural biopsy was taken and 1.6 L of straw-coloured fluid was obtained via a chest drain. The fluid contained glucose 1.3 mmol/L, lactate dehydrogenase 2163 IU/L, protein 49 g/L; no malignant cells were identified. A definitive diagnosis was obtained from the pleural biopsy (Figure 1). Several acid-fast bacilli were identified, and culture grew a fully sensitive *M. tuberculosis*.

Chest Unit, St George's Hospital, London SW17 0QT; [1]Department of Respiratory Medicine, St Helier Hospital, Carshalton SM5 1AA, UK

Correspondence: Dr T B L Ho

E-mail: t.ho@imperial.ac.uk

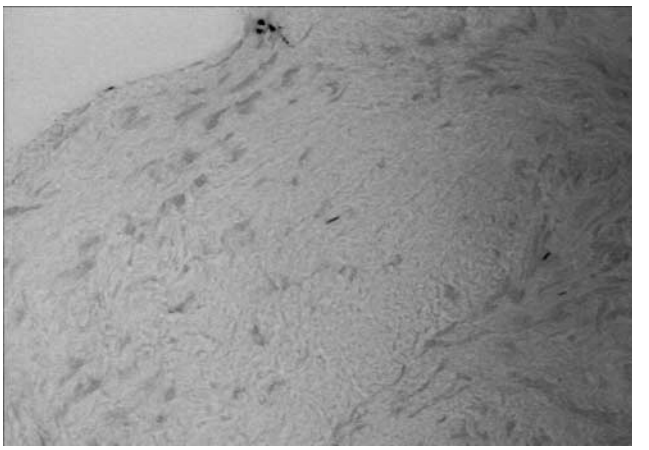


*Figure 1* **Pleural biopsy specimen fixed with Ziehl–Neelsen reagent.** Rod-shaped acid-fast bacillus visible in centre

The patient was then started on isoniazid, rifampicin, pyrazinamide and ethambutol without incident.

## COMMENT

Tuberculosis (TB) remains a global threat to human health,[1] and this diagnosis was considered throughout the patient's admission. However, several investigations for *M. tuberculosis* were negative. This is not unusual in pleural tuberculosis. Acid-fast bacilli were not identified in the pleural aspirate; but, unlike tuberculous empyemas, TB pleural effusions are probably generated by a delayed hypersensitivity reaction to mycobacterial antigens secreted into the pleural space rather than to the bacillus itself.[2] The relative non-reactivity to the Heaf test is also not unusual, with negative tests reported in up to 31% of patients.[3] Co-infection with HIV may also attenuate this test, but our patient was HIV-negative at the time of testing. PCR amplification assays for *M. tuberculosis* can provide a rapid diagnosis and estimation of drug sensitivities.[4] However, reduced sensitivity in pleural disease is thought to be due to the presence of natural inhibitors of the PCR amplification process.[4] Pleural biopsy with both histological and microbiological examination has been reported to have sensitivities of greater than 90% for this condition.[5]

In patients with pleural disease, the presence of negative first-line investigations for tuberculosis should not exclude this diagnosis. A pleural biopsy should be considered.

## REFERENCES

1 Dye C, Scheele S, Dolin P, Pathania V, Raviglione MC. Consensus statement. Global burden of tuberculosis: estimated incidence, prevalence, and mortality by country. WHO Global Surveillance and Monitoring Project. *JAMA* 1999;**282**:677

2 Leibowitz S, Kennedy L, Lessof MH. The tuberculin reaction in the pleural cavity and its suppression by antilymphocyte serum. *Br J Exp Pathol* 1973;**54**:152–62

3 Berger HW, Mejia E. Tuberculous pleurisy. *Chest* 1973;**63**:88–92